Some Relationships Between Heart Attacks and Paralytic Strokes

CLARENCE W. OLSEN, M.D., *Beverly Hills*

SUMMARY

When a patient suddenly collapses and loses consciousness, it is reasonable to assume that both the heart and the brain are acutely disordered. In as many as one case in three in which permanent paralysis occurs, cerebral softening rather than hemorrhage is observed at autopsy. Because there is no proof that in the early stages of stroke the changes are irreversible, and because in a significant proportion of cases the heart is simultaneously injured, both stellate ganglion block and anticoagulant therapy seem justified.

ABOUT a hundred years ago Stokes[11] was collecting and publishing observations on cases of slow pulse. He described what were then called apoplectic, pseudo-apoplectic or syncopal attacks, in which patients suddenly lost consciousness and almost always recovered fairly rapidly and without residual paralysis. He wrote: "The opinion that the apoplectic seizures are owing to deficient arterial supply seems the most tenable. The suddenness of the attack, and, in many instances, the rapidity of the recovery, are in favor of this view."

Hemiplegia with cerebral softening, due to spontaneous or induced bradycardia, has been reported. In one of the cases collected by Stokes, extensive softening of the brain, especially the left cerebral hemisphere, and pathological changes in the heart and aorta, were noted at autopsy. Steegmann and Feil[10] reported two cases of hemiplegia. One patient had auriculoventricular rhythm, with a rate of 32 per minute, and the other had complete auriculoventricular block with a ventricular rate as low as 20 per minute when the pulse was barely perceptible. Steegmann and Feil did not believe that the lesions could be caused by generalized slowing or stoppage of the flow of blood. However, in the second of the two cases, in which death occurred more than a year after the stroke, no arterial occlusion or source of embolism was observed at autopsy.

New possibilities for therapy in cases of cerebral vascular accident provide an incentive for closer analysis of the pathogenesis of cerebral lesions accompanying apoplexy. If there is such a thing as a paralytic stroke that is not necessarily due to hemorrhage, embolism or thrombosis, and in which there is strong tendency toward spontaneous recovery, the fact should be worth recognition.

Of all the cases of cerebral vascular accident, the most dramatic are those in which sudden loss of consciousness occurs. This is not, in all cases, due to hemorrhage. Aring and Merritt[2] noted, in a study of 245 cases, that while coma developed early in 51 per cent of cases of hemorrhage, it also was an early symptom in 32 per cent of cases of thrombosis. Cadwalader[4] observed that in 35 cases of cerebral vascular accident, each of which began with sudden profound loss of consciousness, hemorrhage occurred in 24 and softening in 11.

It should be borne in mind that it is common practice to use the term *thrombosis* rather loosely when it is known only that the lesion in question is an infarct. The term is also commonly applied in cases of infarction of the heart, which are diagnosed as coronary thrombosis although it is known that thrombosis is not essential to myocardial infarction. With this reservation in mind it can be said, on the basis of the statistics just cited, that in as many as one of three cases of infarction of the brain sudden loss of consciousness occurs, and that in the 35 cases of paralytic stroke with sudden loss of consciousness, approximately one in three was caused by cerebral infarction rather than hemorrhage.

In cases of paralytic stroke the presence of a gross lesion of the brain, suspected or verified, may influence one to attribute all the associated symptoms, including loss of consciousness, to the gross lesion. This attitude results from the deep impression made by the first discovery of hemorrhage, thrombosis and embolism in cases of apoplexy. It has only recently been recognized that unconsciousness occurs only in the presence of the following conditions: (1) a lesion of the midbrain, (2) a neighboring or distant intracranial lesion which indirectly disturbs the midbrain, or (3) a generalized depression of function of the cerebrum and brain stem due to circulatory, metabolic or neurotrophic disturbance. Unconsciousness of the kind caused by the third condition mentioned is, when transitory, equivalent to syncope.

COINCIDENCE OF STROKE AND CARDIOVASCULAR INSUFFICIENCY

Investigation has shown quite conclusively that there is a close relationship not only between cerebral vascular disease and cardiovascular disease in general but also between acute myocardial infarction and acute cerebral vascular accident in particular.

Presented before the Section on Psychiatry and Neurology, at the 80th Annual Session of the California Medical Association, Los Angeles, May 13-16, 1951.

Wilson and co-workers[13] recently published observations in a series of 542 cases of acute cerebral vascular accident in which autopsy was carried out in the Laboratory of Neuropathology at the Philadelphia General Hospital. In 361 (66 per cent) of these cases there was either clinical or postmortem evidence of cardiovascular insufficiency at the time of the stroke. In 75 cases an acute cardiac catastrophe was associated with the stroke—acute myocardial infarction in 55, mural thrombus or thrombosis of the cardiac atrium in 12, thrombosis of the aorta or mesenteric arteries in three and rupture of aortic aneurysm in three.

Race and Lisa[7] reported that in 100 consecutive autopsies in which acute myocardial infarction or an acute cerebral vascular accident was observed, both lesions were present in 15 cases. It has been recognized and generally accepted that emotional excitement predisposes both to heart attacks and to paralytic strokes. In this connection, some facts bearing on the controversial subject of cerebral vascular spasm deserve mention. According to Schmidt,[9] constriction is the natural tendency of cerebral blood vessels, when not acted upon by intravascular pressure or chemical vasodilator influences. Carbon dioxide is the best vasodilator of the brain, and acapnea is a potent cause of cerebral vasoconstriction. Thompson[12] observed that in some emotionally excited patients anginal pain accompanied by inversion of T waves or ST segment depression with pronounced lowering of T waves in the electrocardiogram is associated with hyperventilation. Thompson cited Barach who noted that under certain conditions carbon dioxide protected against constriction of the coronary vessels with coronary insufficiency, as observed electrocardiographically.

It is interesting to note that in one patient at the Los Angeles County Hospital for treatment of heart block, asystole was readily induced by voluntary hyperventilation. Syncope with electroencephalographic changes likewise occurred, but the patient recovered from the hazardous experiment without any evident residual effects. It seems likely, however, that emotional discharges can cause simultaneous depression of cardiac and cerebral function, and can give rise to permanent residual effects in the heart and the brain, even though the subject may go through many crises without any permanent ill effect. Engel,[5] discussing syncope in relation to heart disease, made the following comments: "Physicians and patients commonly associate fainting with heart trouble, but most patients who faint do not have heart disease and of those who do, relatively few faint. On the other hand, since heart disease is the most common cause of sudden death, perhaps it would be more correct to say that fainting with heart disease actually is common, but the first attack is usually fatal. Indeed, most of the patients with heart disease who faint eventually die during such an experience."

While Stokes[11] spoke of the apoplexy associated with bradycardia as "non-paralytic," in a sense this is not true. The patient in a syncopal attack is not only unconscious; he is also totally paralyzed so far as any voluntary muscular activity is concerned. The paralysis is, however, usually transient and on recovery nothing in the nature of hemiplegia is observed. In some cases recovery is delayed, and in some of these cases there is a stage at which hemiparesis is observed; but it disappears after a short time. In such cases an inaccurate diagnosis of "transient thrombosis" is sometimes made. If the hemiparesis or hemiplegia following a syncopal attack does not disappear, it is usually assumed that hemorrhage, thrombosis or embolism has occurred, but the concept that generalized failure of circulation to the brain or to the heart can result in focal infarction is a natural conclusion from the foregoing observations. To some extent treatment may avert irreversible changes—hemorrhage, thrombosis or embolism.

As Stokes[11] said, the rapidity of recovery in many instances of syncope with severe heart disease is in favor of the view that the loss of consciousness results from deficient arterial supply to the brain. The object of treatment in cases in which recovery is delayed or imperfect is to encourage return of circulation, and to prevent irreversible changes, particularly thrombosis.

STELLATE GANGLION BLOCK

The effect of stellate ganglion block and anticoagulant therapy, advocated by Aring[3] and others, is a controversial subject. What is still lacking is sufficient controlled study. The good results reported by Amyes and Perry[1] are instructive but not conclusive without a control group. Ruben[8] reported that the death rate in patients admitted to the neurology wards of the Philadelphia General Hospital before stellate ganglion block was used in 100 unselected cases was 82 per cent. In the treated series the death rate was 35 per cent. In a given month when this therapy was given in alternate cases the death rate in the treated group was 44 per cent as compared with 76 per cent in the cases in which stellate ganglion block was not used. Belief that acute cerebral vascular accidents are quite likely to be associated with acute myocardial infarctions would seem to make logical the practice suggested by Aring,[3] namely, employing both cervical sympathetic blocks and anticoagulants in non-hemorrhagic cases, for thrombosis is the most important irreversible complication of infarction of the brain as well as of the heart. Perry and Amyes[6] reported an interesting experience which indicated that stellate ganglion block may benefit the heart and the brain simultaneously. A 65-year-old patient had relief of pectoral pain and of motor aphasia within 10 to 20 minutes after infiltration of the cervical sympathetic trunk with procaine.

9629 Brighton Way.

REFERENCES

1. Amyes, E. W., and Perry, S. M.: Stellate ganglion block in the treatment of acute cerebral thrombosis and embolism, J.A.M.A., 143:15-20, Jan. 7, 1950.

2. Aring, C. D., and Merritt, H. H.: Differential diagnosis between cerebral hemorrhage and cerebral thrombosis. A clinical and pathologic study of 245 cases, Arch. Int. Med., 56:435-456, Sept. 1935.

3. Aring, C. D.: The early treatment of stroke, Cincinnati J. Med., 30:304-309, June 1949.

4. Cadwalader, W. B.: A comparison of the onset and character of the apoplexy caused by cerebral hemorrhage and by vascular occlusion, J.A.M.A., 62:1385-1389, May 2, 1914.

5. Engel, G. L.: Fainting. Physiological and Psychological Considerations, Springfield, C. C. Thomas, 1950.

6. Perry, S. M., and Amyes, E. W.: Stellate block for the relief of simultaneous angina pectoris and motor aphasia, California Med., 71:422-423, Dec. 1949.

7. Race, G. A., and Lisa, J. R.: Combined acute vascular lesions of the brain and heart, Am. J. M. Sc., 210:732-737, Dec. 1943.

8. Ruben, J. E.: Results of stellate ganglion block therapy for cerebro-vascular accident, J. Philadelphia Gen. Hosp., 1:110-115, Oct. 1950.

9. Schmidt, C. F.: The Cerebral Circulation in Health and Disease, Springfield, C. C. Thomas, 1950.

10. Steegmann, A. T., and Feil, H.: Hemiplegia following bradycardia and cardiac standstill, Am. Heart J., 25:244-230, Feb. 1943.

11. Stokes, W.: Observations on some cases of permanently slow pulse, Dublin Quart. J. M. Sc., 2:73-85, 1846. Reprinted in M. Classics 3:727-738, March 1939.

12. Thompson, W. P.: Electrocardiograph in hyperventilation syndrome, Am. Heart. J., 25:372-390, March 1943.

13. Wilson, G., Rupp, C., Jr., Riggs, H. E., and Wilson, W. W.: Factors influencing the development of cerebral vascular accidents. I. Role of cardiocirculatory insufficiency, J.A.M.A., 145:1227-1229, April 21, 1951.